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Development of a model to demonstrate the effects of friction and pressure on skin in relation to pressure ulcer formation

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ABSTRACT

Pressure ulcers are a common injury of the skin which leads to pain and potential infection for patients and financial burden to the healthcare providers across the global due to treatment costs, litigation and extended hospital stays. The current study focuses on one of the causes of pressure ulcer formation, ischemia. Blood vessels are deemed to be deformed and blood flow restricted when skin is subjected to external mechanical loads including friction, pressure and the combination of both. Hence, normal oxygen delivery to cells or metabolic waste removal are locally stopped which causes cells deaths and ultimately pressure ulcers.

The current study proposes a 3D finite element analysis model which is capable of demonstrating the effect of friction, pressure and the combination of both to the deformation of blood vessels. The results of simulation suggested that applied pressure collapsed the blood vessels while friction opened up the blood vessels. However, as a combination effect of pressure and friction, the cross-sectional areas of blood vessels were reduced significantly. This model is clinically and physiologically relevant in terms of loading regime and blood vessels structures. The model with further development can be adopted to be an effective tool to evaluate the effects of medical devices to the possibility of pressure ulcer formation.

1. Introduction

The aim of the study is to develop a Finite element analysis (FEA) model to predict the effect of friction and pressure on skin in relation to pressure ulcer (PU) formation. There are two main reasons which are known to increase the possibility of PU formation; ischemia and excessive amounts of internal strain resulting in deep tissue injury [1,2]. The current study focuses on the condition of ischemia resulting from external friction and pressure. Skin and soft tissue becomes distorted when external friction and/ or pressure are applied to skin surface, this results in compression of the blood vessels. This phenomenon has a physiological effect restricting blood flow, with the restriction in blood flow, perfusion of oxygen is limited and removal of metabolic waste is inhibited which leads to cells death in the affected area resulting in ischemia followed by PU formation [3,4]. The proposed model in the current study demonstrates the effect of friction and pressure to the reduction of cross-sectional areas of blood vessels in the different layers of skin including the dermis and hypodermis. This initial model is simplified and the skin is modelled to be isotropic and non-viscoelastic. These initial simplified models are clinically and biologically relevant in terms of skin and blood vessels structures. The loading conditions have been developed by conducting pressure mapping of support surfaces with healthy volunteers in a clinical setting to ensure validity of the loading regimes used. It is proposed that this model with further development could be used to predict the effects of medical devices on the change in blood vessel dimensions whilst indicating the probability of PU formation.

Skin is the biggest organ and often not given the attention that it deserves as one of the most important. It serves to protect the internal organs, prohibit external hazards affecting the body, regulate temperature and absorb shocks. The stratum corneum, epidermis, dermis and hypodermis/ subcutaneous fat are the key layers of the skin Fig. 1. Each of these layers is unique, with different mechanical properties and varying distribution and configuration of blood vessels. This versatile organ can be subjected to various injuries and trauma, one severe instance which causes loss of integrity in the skin as a barrier to bacteria and infection is pressure Ulcers (PU) and friction blister formation.

Pressure ulcers are a skin injury that used to be labelled as “bedsores” or “pressure sores”. They can be just a minor

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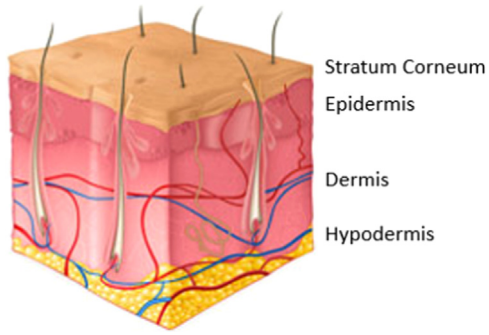

Fig. 1. Diagram of skin structure [5].

discolouration to the skin or potentially life threatening full thickness skin damage affecting all layers from the epidermis to the muscles or even through to the bone. PU usually occur at areas of bony prominence, for instance, the elbow, heel and back of skull. Multiple mechanical parameters including friction, shear stress, pressure and microclimate are believed to be included in the potential triggering factors [6,7].

An increasing trend of PU formation is a financial burden to the healthcare systems across the globe. Treatment cost of pressure ulcers increased from USD 1.3 billion in 1998 to USD 11 billion annually in 2014 in the United States [8–10]. The yearly treatment cost for PU was £1.4 – 2.1 billion in the UK and USD 1.6 billion in Australia [11,12]. The mean cost of treating a pressure ulcer is estimated as £1,214 for stage one ulcer up to £14,108 for stage four [13]. It is more cost effective to prevent the occurrence of ulcers than provide treatment especially for the stage four ulcer [14,15]. According to the NHS information, "Under half a million people in the UK would develop at least one pressure ulcer in any given year" [16]. 11% of the total residents in US nursing homes developed pressure ulcers [17]. These statistics alone demonstrate the urgent need to provide information and development tools which can help clinicians address this global issue.

Furthermore; pressure ulcers are not only a financial burden to the healthcare system but also have the potential for serious impact to patients' health. The increasing trend of pressure ulcers was shown in the study conducted by Russo in 2008. There was only a 15 percent increase in total numbers of hospitalisation throughout the population. However, the number of pressure ulcers developed during the hospitalisation increased 78.9 percent from 281,300 to 503,300 from 1993 to 2006 in the United States [18]. Patients were shown to be restricted and limited in their social and leisure activities and psychologically impaired, especially for prolonged ulceration over six months in duration. Pain from PU was always neglected or unrecognised while pressure-relieving mattresses and cushions were reported to be uncomfortable, too hot, and noisy. Patient's quality of life was lowered by all these factors [19]. Worse still, PU can be fatal and life-threatening in some cases where cellulitis, blood poisoning, bone and joint infection, necrotising fasciitis and gas gangrene developed as a result of PU formation [20]. All these are the possible consequences of untreated stage 3 or 4 pressure ulcers.

The pressure ulcers occur usually as a result of self-weight, for instance, the weight of foot acting on the heel. However, another type of pressure ulcers are labelled as medical device related (MDR) pressure ulcers is of growing concern. Hospitalised patients often require medical devices or other interventions to treat or monitor their physiological conditions. These devices are usually attached to the patient's body with tapes or other fixing methods in order to function correctly. Pressure is often applied to the skin as a result of these fixation methods and some patients cannot even detect discomfort due to unconsciousness, drug therapy or lack of sensations. The medical device on the skin also acts like tourniquet which restricts the blood flow and increases the possibility of pressure ulcers formation [20]. There is also the potential for heat and moisture development at the skin-device interface, this is often referred to as the micro-climate, this refers to the environment and conditions in the surrounding area of direct contact. Out of the total number of pressure ulcers, one third of them were shown to be related to medical devices in a study conducted by Black [21].

A number of studies have been conducted by other authors investigating modelling as a tool to investigate the behaviour of skin incorporating external mechanical or biological factors and other skin injuries [22–24]. However there is little evidence to show the use of three dimensional computational modelling of skin incorporating the structures of blood vessels to demonstrate the effect of mechanical loading typically friction and pressure to PU formation.

2. Method

Finite element analysis (FEA) is a conventional computational method of demonstrating stress, strain and deformation of a model in various disciplines. FEA has been adopted as the analysis method for the behaviour and deformation of skin and blood vessels under friction and pressure, the primary aim being to investigate the use of FEA in the prediction of pressure ulcer formation. Abaqus CAE 6.14 was utilised to conduct the simulation.

In a study by Xu in 2011 FEA was used to simulate the material deformation following nano-indentation of a large block of material, there are some similarities in this method with the study of pressure ulcer formation as the loading contributes gradually less effect on the surrounding are moving away from the point of peak pressure [25]. In the current study the method is focussed less on the deformation of the skin layers and more on the reduction in cross sectional area of the blood vessels within the layers of skin with a view to investigating the reduction in potential blood flow in the areas of high pressure.

2.1. Features of the model

For the purposes of modelling the deformation in blood vessels, the model was created in the micrometre scale. The skin demonstrated by the computational model was circular, however the computational model was presented as a quarter as it was symmetrical in X and Z directions. The model was partitioned into 9 unevenly spaced radial sections with the total radius of 20,600 μm as shown in Fig. 2(a). These sections are label as Section 1 to 9 from centre to the outermost section. The important feature of the model was the blood vessels located at the centre of the model, and the boundary of the model was designed to be a large distance away from the centre in order to have minimum effect to the deformation of affecting the blood vessels in the central region. Iterative simulations demonstrated that 20,600 μm was an appropriate dimension for the boundary conditions. The stratum corneum, epidermis, dermis and hypodermis were the four vertical layers of skin modelled as shown in Fig. 2(b).

2.2. Internal features (Blood vessels) of the blood

Nine horizontal blood vessels were constructed inside the centre section of the model. Two vessels were located at the dermis layer and one of those was near the epidermis while another one was near the dermis–hypodermis interface as suggested by Michel Démarchez [26]. Démarchez also mentioned that these two vessels were connected by vertical vessels which transfers blood from the deeper part of the skin to the cells located at epidermis and dermis layer. All the blood vessels are similar but slightly

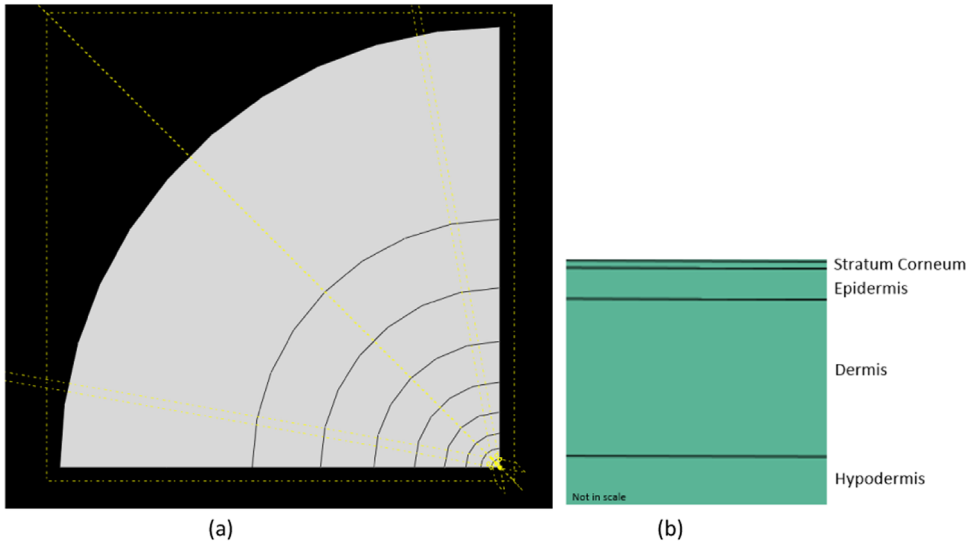

Fig. 2. Features of the model.

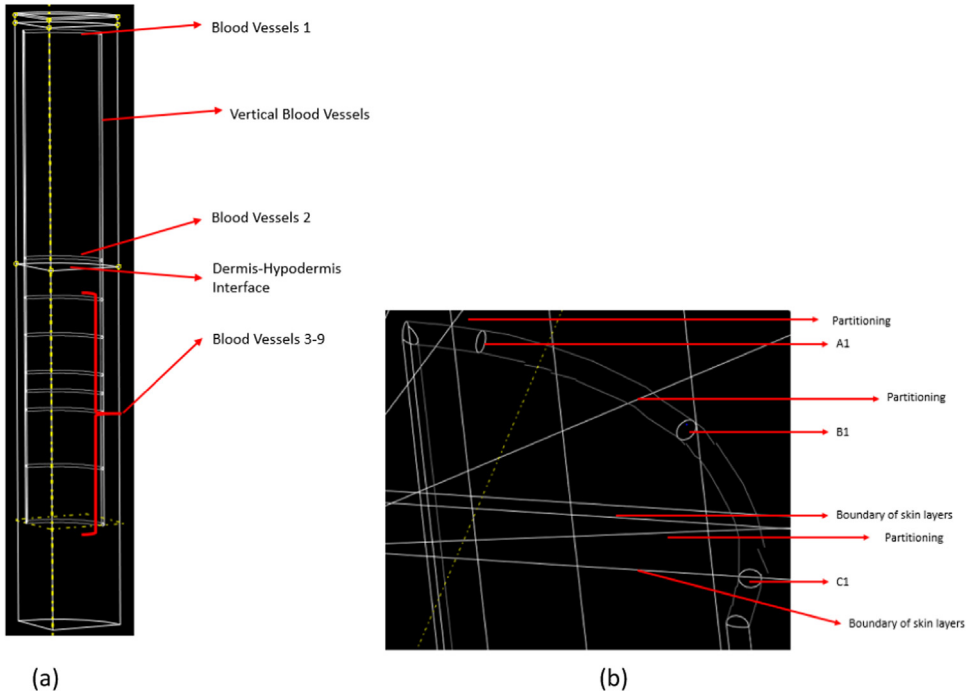

Fig. 3. Internal features (blood vessels) at the centre, (a) blood vessels distribution (b) locations A1, B1 and C1.

bigger than capillaries, those created in the current model were 15 μm. The hypodermis is highly vascularised according to Saladin [27], 7 horizontal blood vessels were created at the hypodermis layer with random vertical spacing, Fig. 3(a). The blood vessels structure in this initial model was simplified, they would typically be of a more complex shape in reality. The positions were referenced to previous studies conducted by other authors [28,29]. Although the blood vessels would typically be distributed across the entire skin, the model only represents those at the centre of the loading area. On each of the horizontal vessels, there were 3 locations where the cross-sectional areas were analysed which were labelled as A, B and C followed by the vessels number 1–9 as shown in Fig. 3(b). The intersecting lines from Fig. 3(b) were partitioning lines and also the boundaries of different layers of skin as labelled on the figure. The partitioning ensuring nodes were allocated at the intersecting faces of the partitions and blood vessels. Fig. 3(b).

2.3. Mechanical properties and thicknesses of skin layers

It is very difficult to determine the mechanical properties of skin, this is due to the fact that the properties are not stable, skin changes due to age, nutrition, condition and hydration amongst other things. A number of authors have investigated the properties of various layers of skin, these studies have been conducted on both human and animal samples. From the literature the most representative properties were selected and used in this model. The development of this model will require an accurate method of determination of mechanical properties at the appropriate scale for inclusion in the modelling. Table 1 shows the list of dimensional and mechanical properties which were used and incorporated into the model.

Table 1
Mechanical properties and thicknesses of skin layers.

Dimension/Parameter		Value	Reference
Thickness of	Stratum corneum	10 μm	[30,31]
	Epidermis	42 μm	
	Dermis	1,285 μm	
	Hypodermis	1,913 μm	
Young's modulus of	Stratum corneum	1 MPa	[30]
	Epidermis	0.05 MPa	
	Dermis	0.6 MkPa	
	Hypodermis	0.11 MPa	
Poisson's ratio of skin		0.5	[32]

2.4. Boundary condition

Accurate and representative boundary conditions greatly affect the accuracy of an FEA simulation. In order to ensure efficacy of the model, 2 types of boundary condition were applied in the model. These were rigid and symmetrical boundary conditions. Fig. 4 shows the configuration of the boundary conditions.

By using the symmetrical boundary conditions, the model was sectioned into a quarter to enable faster simulation run time and increased mesh density.

The bottom plane of the model would be attached to muscle in reality, however an immovable boundary condition was applied. Muscle in real life is stiffer than skin which tends to have less deformability and its stiffness changes vigorously with use. It would be too complex in this initial model to include the behaviour of muscle and it was felt that it would not have a massive influence on the reduction of cross-sectional areas of blood vessels.

2.5. Loading and loading area

Friction and pressure were applied to the model on the top surface. The size of the loading area did affect the deformation of the interested area of the skin model which was the centre sections. A pressure of 38.6 kPa was applied at the top surface of the centre section at simulation test runs. This magnitude of pressure is reported to be the pressure required to cause a PU without the presence of friction [33]. The loading area was determined by analysing the effect of size of area on the changes in deformation of the blood vessels. Separate simulations were conducted to determine the effects of pressure only, friction only and pressure with friction on cross sectional area of blood vessels. The final findings of the appropriate loading area was presented in the results section under the sub-section of simulation test run.

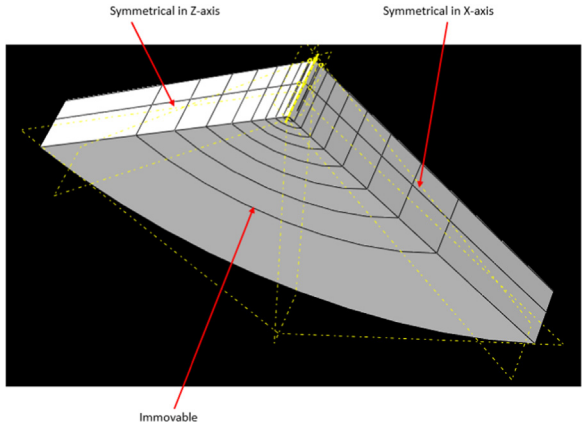

Fig. 4. Application of boundary condition.

2.6. Meshing

Tetrahedral and hexahedral elements were adopted for the centre section and the rest of the model respectively. Due to the complicated geometries at the centre, it was impossible to partition all the geometries to apply hexahedral elements. The interested section of the model was fairly small as compared with the whole model. In order to run the simulation effectively and efficiently, the size of the elements were increased further away from the centre. Incompatible contact were found between the contacting surfaces of two types of elements. Consequently, automatic tie constrains had to be applied.

2.7. Pressure mapping

Pressure mapping was an efficient way to evaluate the pressure between the patients and the supporting surfaces. A TEKSCAN CONFORM at pressure mapping system was adopted for acquiring the peak pressure between 3 healthy volunteers and the rigid and soft support surfaces. The volunteers sat on the support surfaces with vertical upper body. The peak pressure from this experiment was applied in the computational model to demonstrate the effect of the pressure to the blood vessels.

2.8. Friction calculation

Friction was applied to the surface of model to demonstrate the effect of friction to the blood vessels with and without the present of pressure. The amount of friction was calculated by **Error! Reference source not found.** where F_r, μ and N are the magnitude of friction, coefficient of friction respectively. The coefficient of friction adopted for acquiring friction in this study was 0.79 which was the largest value found in a study conducted by Vilhena published in 2016 in order to simulate the highest frictional situation [34].

$$F_r = \mu N \quad (1)$$

3. Results

3.1. Pressure mapping

3 volunteers with the BMI of 19.9, 26 and 30 took part in the pressure mapping experiment. The results were listed in Table 2. The highest peak pressure was found when volunteer 1 was supported by a rigid surface. This magnitude of pressure was further applied to the computational model to demonstrate the effect to the deformation of blood vessels.

Table 2
Table of peak pressure acquired from pressure mapping for 3 volunteers.

Supporting surface	Volunteers(BMI)		
	Volunteer 1 (19.9)	Volunteer 2 (26)	Volunteer 3 (30)
Rigid	89 mmHg	89 mmHg	51 mmHg
Foam	63 mmHg	60 mmHg	28 mmHg

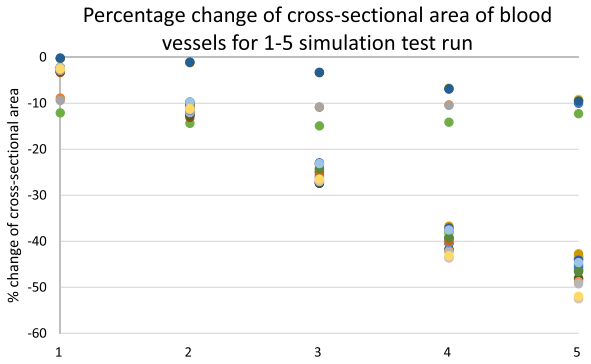

Fig. 5. Graph of percentage change of cross-sectional area of blood vessels for 1–5 simulation test run. The data points represent various locations on the model. 1: loading at centre only; 2: loading at Sections 1 and 2; 3: loading at Sections 1–3; 4: loading Sections 1–4; 5: loading at Sections 1–5.

3.2. Simulation test run

Five Simulation test runs were conducted to analyse the relationship between the loading areas of skin and the deformation of blood vessels. The deformations of blood vessels were significantly higher at the simulation test runs with larger loading areas as demonstrated in Fig. 5. The loading area of test run 1 was only at Section 1 while 2 was at Sections 1 and 2 and so on. The percentage change of the cross-sectional area of the blood vessels were plotted in Fig. 5. The data point at test run 5 could be regarded as two main groups, the less percentage change group and higher percentage change group. It was recognised by cross-checking the data from the simulation that the less deformed group was the blood vessels that located at dermis layers while the more reduction data was from the vessels at hypodermis layer. This was further discussed in discussion.

3.3. Calculated friction

The magnitude of friction that was applied to the computational model was deduced from **Error! Reference source not found**. As the centre of the loading area was pushed vertically downward, only the surrounding areas would be affected by the friction. Thus, the average pressure surrounding the peak pressure area were 35 mmHg (4666.3 Pa) and further converted to be the vertical force for Equation 1. The calculated magnitude of friction per unit area was 3499.7 N/m2 and applied to the model.

3.4. Effect to reduction in cross-sectional area of blood vessels

Three scenarios were simulated including pressure alone, pressure with friction and friction alone. The percentage of reduction in cross-sectional area is shown in Fig. 6. It is important to note that A1–C2 sections were located in the dermis layers of the skin while the rest of the sections were located in the hypodermis

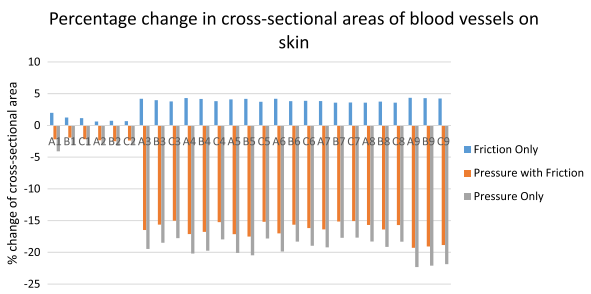

Fig. 6. Graph of percentage change in cross-sectional areas of blood vessels on skin.

layers. The blue, orange and grey bars represented the percentage change of the cross-sectional areas of the blood vessels. The results are further analysed in the discussion.

4. Discussion

There was a significant decrease in cross-sectional area of the blood vessels from simulation test run 1–4 in which loading was applied at section 1, 1 and 2, 1–3 and 1–4 respectively. The percentage change in the cross-sectional area tended to be stable at test run 5 as shown in Fig. 5. Thus, 5 sections were loaded for the actual simulations to demonstrate the effect of pressure alone, friction alone and pressure with friction to the deformation of blood vessels. Fig. 5 also showed that the vessels located in the dermis layers had less reduction in cross-sectional area as compared with those located at hypodermis layer. This could possibly due to the difference in mechanical properties of the dermis and hypodermis layers. It also demonstrated that the reduction in cross-sectional area was more significant for the vessels which were located vertically closer to the bottom surface than those near the dermis layer.

Previous knowledge suggested that less amount of pressure is required to cause a PU if combined with friction forces than without friction [33]. In other words, friction as well as pressure causes reduction in cross-sectional area of blood vessels. However, in the currently study in which a sitting posture was simulated, the effect of friction to blood vessels was increasing the cross-sectional areas of blood vessels rather than reducing. It was consistent with the fact that radial inward force was applied to the supporting surface, however in reality the bone and the body weight was pushing the skin downward to the deforming foam (supporting surface). Consequently, the foam/ textile applied opposite direction frictional force to the surface of skin radial outward. This radial outward force caused the skin in different layers to be pulled outward, hence, the cross-sectional areas of the blood vessels located in the skin was increased. This effect was demonstrated in the results (Fig. 6). In contrast to the effect of friction, the effect of pressure to blood vessels was predictable, reducing the cross-sectional areas of blood vessels suggested by the trend shown in Fig. 6. Although friction showed an increase in cross sectional area, the combination effects of pressure and friction showed less reduction in cross sectional area however it was still evident.

This was in contradiction to clinical evidence and highlights one of the limitations of the model that for the sitting posture used in this simulation, the friction and pressure do not function in the same was as that seen in bed ridden patients. This also highlights the need for a different approach into investigating device related pressure injury and those pressure ulcers caused by long periods of immobility.

Pressure ulcers in most cases started from superficial layers. It was assumed that ischemia started at top layers as well. However, the simulation results demonstrated that the cross-sectional area of blood vessels in hypodermis layers were significantly more reduced as compared to those in dermis layer. Furthermore, the blood is transferred through the blood vessels in hypodermis layer to the dermis layers. As a result, the blood flow in dermis layers would be affected if the blood flow in hypodermis was restricted.

This proposing model demonstrated the ability to help to understand the effect of change in blood vessels. Different magnitudes of pressure and friction can be applied to the model to demonstrate different postures of patients for instance, sliding down an inclining bed, being dragged up from a low position, a device which has rotation or sliding forces interfacing with skin etc.

The model was assumed to be non-viscoelastic, linear and homogeneous which limits the ability of showing the effects of

loading depending on time and the effect of non-linear and inhomogeneous. However, this model was an initial step towards modelling the effect of blood vessels located inside skin by a 3D FEA model. The model can demonstrate the immediate effect of both pressure and friction to blood vessels which is a consideration factor for medical device development. This model simplified the geometry of blood vessels and ignoring the presence of capillary. It would be very complicated to model all different types of vessels in the same model and the success of the current model is a very important method to model other sizes and types of vessels.

5. Conclusion

A 3 dimensional FEA model had been created for demonstrating the effects of pressure, friction and combination of both to the deformation of blood vessels at different layers of skin. Simulation results suggested different magnitudes of deformation were found at different vertical locations of blood vessels. A better understanding of the effect of friction of sitting posture to skin was demonstrated by the simulation results. This model is a step toward the success of a complete simulation of mechanical loading to the deformation of blood vessels in skin. A few significant points regarding to the blood vessels located at hypodermis layer demonstrated in the simulation (sitting posture) are listed below:

1. Friction opened up the blood vessels by approximately 3.95%.
2. Pressure reduced the cross-sectional area of blood vessels by about 17.7–22.3%.
3. The combined effect of friction and pressure showed a reduction in the cross-sectional area of blood vessels by about 15.0–19.3%.

6. Further Development

The current model only consisted of blood vessels at the centre part of the loading area. It is essential to understand the deformation of blood vessels at the side of loading area as well. Furthermore, the viscoelastic and non-linear effect of skin are two major directs of further development.

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